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THE NATURE OF THE VASCULAR COMMUNICATIONS BETWEEN THE CORONARY ARTERIES AND THE CHAMBERS OF THE HEART*†‡

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THE existence of direct vascular communications between the coronary arteries and the chambers of the heart has been claimed and denied. Such evidence as has been presented in favor of the existence of these channels has been indirect and based largely upon experiments in which perfusions and injections of the coronary vessels were employed. The use of the same general methods variously modified has led others to deny the existence of channels between the coronary arteries and the heart chambers. If such channels do exist they almost certainly play a rôle in the circulation of the heart, but at the present time there is no agreement as to the nature or the importance of that rôle. It seemed wise, therefore, to study the vessels by injections and by the rather tedious method of serial sections and wax-plate reconstructions in order to establish their anatomical and histological structure. Such knowledge, if established, should be of definite value to those investigators who, in studying the physiology of the coronary circulation, have been compelled to make one of two assumptions; namely, that vascular channels between the coronary arteries and the atria and ventricles either do or do not exist.

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†The results reported in the first part of this paper (celloidin injection experiments) were carried out in 1928 while Dr. Mettier was working with me in the Thorndike Laboratory. The investigation has since been extended and completed in the H. K. Cushing Laboratory where Dr. Klumpp joined in the work. Miss Zschiesche assisted in the investigation throughout. J. T. W.

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It is proposed to present here histological and other evidence of the existence of direct vascular channels between the coronary arteries and the chambers of the heart, as well as the results of certain observations upon the histological nature of these communicating vessels.

The belief that the coronary arteries have direct vascular communications with the chambers of the heart is not a new one. The idea was first advanced by Raymond Vieussens in 1705 in a letter to Monsieur Boudin, Conseiller d'Etat, Premier Medicin de Monseigneur le Dauphin. Boudin's reply in which he acknowledged receipt of Vieussens' letter was dated July 9, 1705. Vieussens' letter was published in 1706.[1] In it he related how, in examining the depth of the roots of a large polyp which had formed in the right ventricle of the heart of a man who had died of a slow fever accompanied by violent palpitation of the heart, he traced the firmest roots of the clot as far as certain holes which seemed to him to be the orifices of specific ducts. He made several similar observations and from them concluded that blood circulating in the medial and inner fleshy vessels of the heart was carried into the cavities by the ducts in which the polyps first take root. Next he injected saffron dye, dissolved in spirits, into the left coronary artery and observed its passage into the left atrium and left ventricle, but none escaped via the veins into the right chambers. He called these communicating vessels "fleshy" ("le reste est poussé en partie dans le ventricule gauche de ce viscere par des vaisseaux, que j'appelerai charnus cy-aprés"). Vieussens also dissected human hearts and the hearts of sheep and calves, and, with the aid of a microscope, found the small openings (ouvertures) in the chambers of all, in some instances with delicate valves over the openings while in others valves were lacking. He noted that many of the channels (conduits) received blood from several fleshy vessels. To these he gave the name "common vessels" and to their orifices the name "common openings" ("je les appelleray des vaisseaux communs: j'appelleray aussi leurs embouchures des ouvertures communes").

These vessels and openings, so clearly described by Vieussens, have since been confused with somewhat similar ones described by Thebesius[2] two years later in 1708, and as a result all such vessels are now commonly known as thebesian veins. Thebesius injected certain substances into the coronary veins and noted their escape into the heart cavities through small openings in the endocardium. He was familiar with Vieussens' work and so states in his paper.

Thebesius' belief that the vessels were connected only with veins was supported by several anatomists of the day and Vieussens' work was soon forgotten. Lancisi[3] in 1740 injected mercury into the coronary arteries and observed its appearance in the heart chambers, but believed with Thebesius that it escaped through venous channels (thebesian veins). In 1798 John Abernethy,[4] by making a "common coarse waxen

injection'' into the coronary arteries, observed that it flowed readily into the cavities of the heart. He also injected masses of a different color into the arteries and veins and concluded that vessels from arteries and veins communicated with the heart chambers ''because the injection which was employed was too coarse to pass from one set of vessels to the other, and yet the different colored injections passed into the cavities of the heart unmixed.''

Then followed a period that might be termed the dark ages for the ''vaisseaux charnus'' and the thebesian veins, but it came to an end with the work of F. H. Pratt[5] in 1898 who, by means of perfusion of defibrinated blood through the ventricle of a cat's heart, kept it beating for an hour. This very important work furnished the first experimental evidence that the thebesian vessels might serve as an entrance for blood from the ventricles into the capillaries.

In 1928, Wearn[6] perfused the coronary arteries of human hearts with India ink and observed that the perfusate for the most part escaped into the chambers of the heart through the thebesian vessels. Histological sections of these hearts revealed the fact that very few, and in many instances scarcely any, of the capillaries contained ink particles. Moreover, when a celloidin mass too thick to pass through capillaries was injected into the coronary arteries, celloidin plugs were found protruding from small openings in the endocardial walls of the atria and ventricles, while the capillary bed remained uninjected. From these experiments the conclusion was drawn that direct vascular communication existed between the coronary arteries and the heart chambers. F. H. Pratt[7] has also observed plugs of gelatin protruding from the thebesian openings after injection of this thick mass into the coronary arteries. Mettier, Zschiesche, and Wearn[8] in 1929, and Wearn, Klumpp, and Zschiesche[9] in 1932 published the preliminary results of some experiments which are reported in full in this paper.

Grant and Viko[10] in 1929 injected the thebesian vessels through their endocardial foramina and through the coronary vessels. They used finely drawn glass cannulas of such sizes as to fit snugly into the endocardial foramina. Their injection pressure was produced by blowing from the mouth into the injection system. By this means a pressure of about 50 to 60 mm. Hg was obtained. They were able to confirm the existence of anastomoses between thebesian vessels and coronary veins and described several types of venous anastomoses. They also injected the coronary arteries but failed to find evidence of direct arterial communications and concluded that ''the coronary arteries communicate with the thebesian vessels only through capillaries.''

Stella[11] in 1931 employed the denervated heart-lung preparation in dogs in an attempt to test the claim of the existence of communications between the arteries and heart cavities by a physiological method. He

was able to maintain the pressure within the ventricles and drop the pressure within the coronary arteries and, since under such conditions he was unable to demonstrate back-flow from the chambers into the arteries, he concluded that his work did not support the existence of large channels connecting the thebesian veins with the coronary arteries.

Grant[12] in 1926 found in a child's heart, which presented a congenital anomaly, persistent channels communicating freely with the heart cavities and with the coronary arteries, veins, and capillaries. Bellet, Gouley, and McMillan[13] in 1933 reported dilated sinusoids or thebesian veins in a heart which showed an advanced tuberculous fibrocaseous infiltration.

In view of the lack of agreement on the existence of vascular connections between the coronary arteries and the heart chambers, the experiments reported in this paper were devised and carried out.

METHODS AND OBSERVATIONS

Human hearts obtained at necropsy were used in the experiments reported in this study. No effort was made to select normal or abnormal hearts. Hearts were accepted for injection whenever they could be obtained from the necropsy table. The causes of death covered a wide range, but it so happened that none of the hearts showed any gross evidence of any pathological process.

The use of thick celloidin as an injection mass offered a method of approach in determining the presence and constancy of arterial communications with the heart chambers. In a previous experiment[6] it was found that celloidin could be prepared in a suspension sufficiently thin to pass through arteries and arterioles but too thick to penetrate the capillaries. The injection mass consisted of a 5 per cent solution of celloidin in acetone. A blue fluid mass was obtained by mixing crystal violet and brilliant green with the celloidin, and a red fluid mass was made by the addition of alkanin to the celloidin.[14] The red celloidin mass was used in making a cast of the ventricular chambers, and the blue for casting the coronary arteries. The following experiment is a typical one and illustrates the method:

PROTOCOL

Heart 10. Male. Aged thirty-two years. Death was the result of staphylococcus septicemia. The heart was placed in the ice box for twenty-four hours to allow for the passing off of rigor mortis, following which it was gently massaged in warm salt solution to break the remaining rigor and to remove all blood clots in the chambers. The coronary sinus and the tricuspid valve were closed by purse-string sutures. (In some instances the valve leaflets were everted and clamped.) A cannula was tied into each coronary artery and into the pulmonary artery. The red celloidin mass was injected through the cannula in the pulmonary artery into the right ventricle at a pressure of 160 mm. Hg, and this pressure was maintained until the experiment was completed. At the same time, suction was applied to the

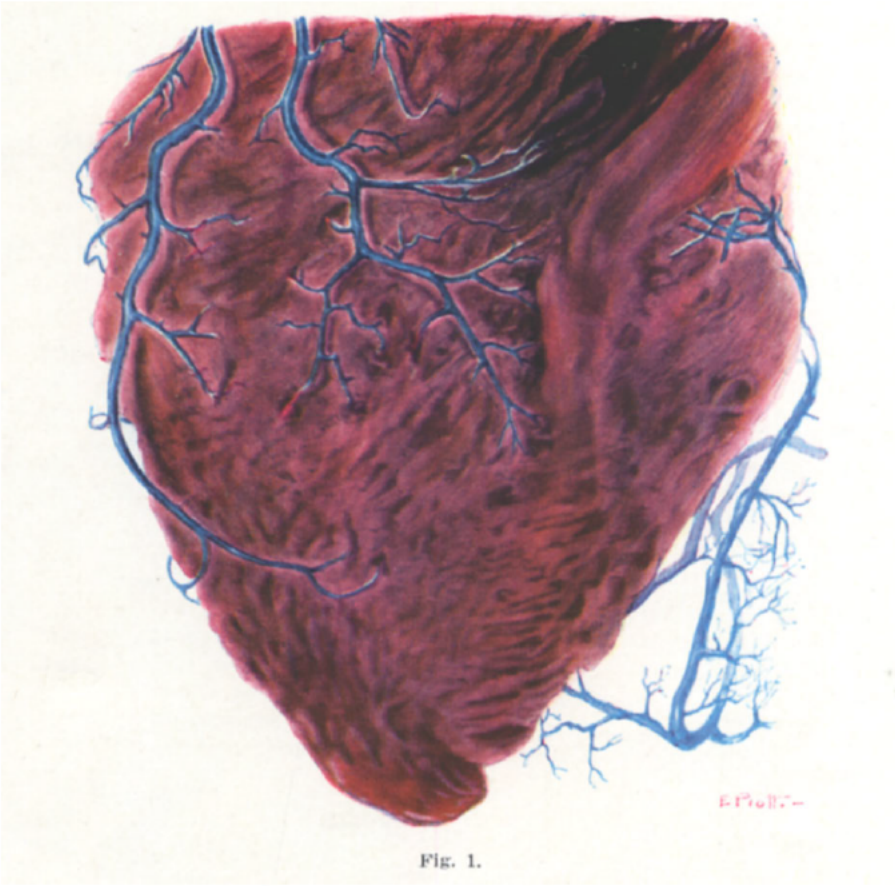


Fig. 1.

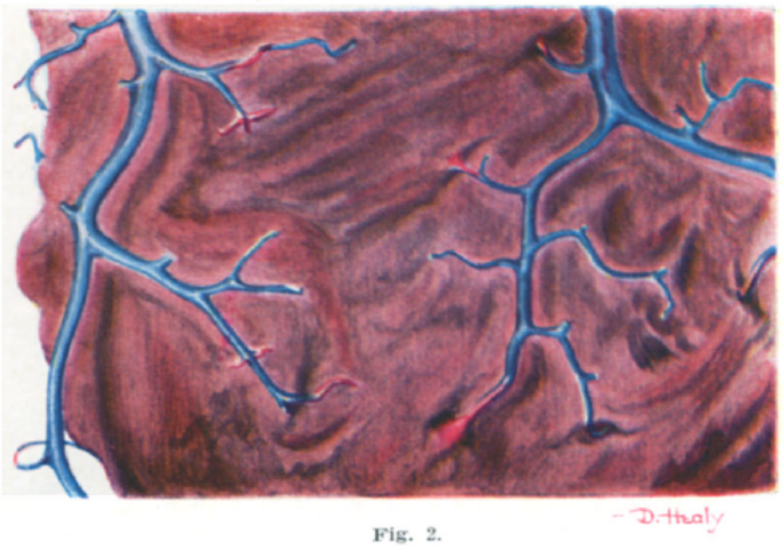


Fig. 2.

Plate 1

Fig. 1.—Heart 10. The painting shows a red celloidin cast of the chamber of the right ventricle and a blue celloidin cast of the coronary arteries. The outer branches of the arteries have been removed in order to show the red tips at the junctions of the coronary arteries and the red cast of the chamber. The artery tips are firmly fused with the celloidin cast of the right ventricular cavity (see Protocol of Heart 10).

Fig. 2.—Heart 10. A painting on a larger scale to show more clearly the red tips at the points where the casts of the coronary artery fuse with the cast of the right ventricular cavity (see Protocol of Heart 10).

cannulas in the coronary arteries. After a lapse of ten minutes the suction was discontinued, care being taken to prevent the entrance of air, and the blue mass was injected into the coronary arteries at a pressure of 180 mm. Hg. This pressure was maintained for fifteen minutes, after which the cannulas were clamped off and the heart was plunged immediately into ice water where it remained for forty-eight hours to permit maceration of the muscle. The heart was then placed in 75 per cent HCl for seventy-two hours, during which time the acid was changed twice. This usually completed maceration of the muscle. The cast was washed free of muscle by playing a very fine stream of water on the débris. (Extreme care was necessary in this procedure to avoid breakage of the finer vessels.) After washing, the cast was mounted.

A reproduction of a painting of this specimen is shown in Plate I, Figs. 1 and 2, and a photograph of the same specimen in Plate II, Fig. 1. The specimen consists of a solid red cast of the chamber of the right ventricle and blue casts of the anterior branches of the right and left coronary arteries. It is to be noted that some of the terminations of the branches of the anterior branch of the right coronary artery are red in color. These red tips vary from 4 to 15 mm. in length. The distal ends are continuous with the red mass in the ventricles, and the proximal ends fuse smoothly with the blue mass in the arteries. Such vessels when grasped between the fingers or with forceps are found to be firmly attached by this red tip to the ventricular mass, showing complete fusion of the two celloidin masses. The cast of the vessels is smooth and round and shows no lateral communications. Capillaries are not injected. It is evident, therefore, that the red celloidin mass has found its way directly from the right ventricle into the terminations of the right coronary artery, as shown by its fusion with the blue mass injected into the artery at its origin. The pressure within the right ventricle probably aided in the entrance of the red mass into the ends of the branches of the coronary arteries.

Fifteen human hearts were injected according to the technic described in the preceding protocol. In all of the final specimens red tipped terminations of arteries fused to the ventricular mass were found. The number of these vessels in each heart varied with the completeness of the injection and with the degree of damage that occurred during the washing process. A composite of all of the specimens injected would average about twenty to twenty-five communicating arteries scattered generally over the heart walls. It is not felt that these figures represent the total number of communications, for in many instances it was obvious that the injection was incomplete.

These communicating vessels between the coronary arteries and the ventricles usually had their origin from the main trunks of the coronary arteries during their course toward the apex of the heart. Many of the deeper branches of the arteries lying near the endocardium also gave off frequent branches. As many as eight to ten communicating branches were frequently seen in the course of the anterior branch of the left

Fig. 1. Fig. 2.

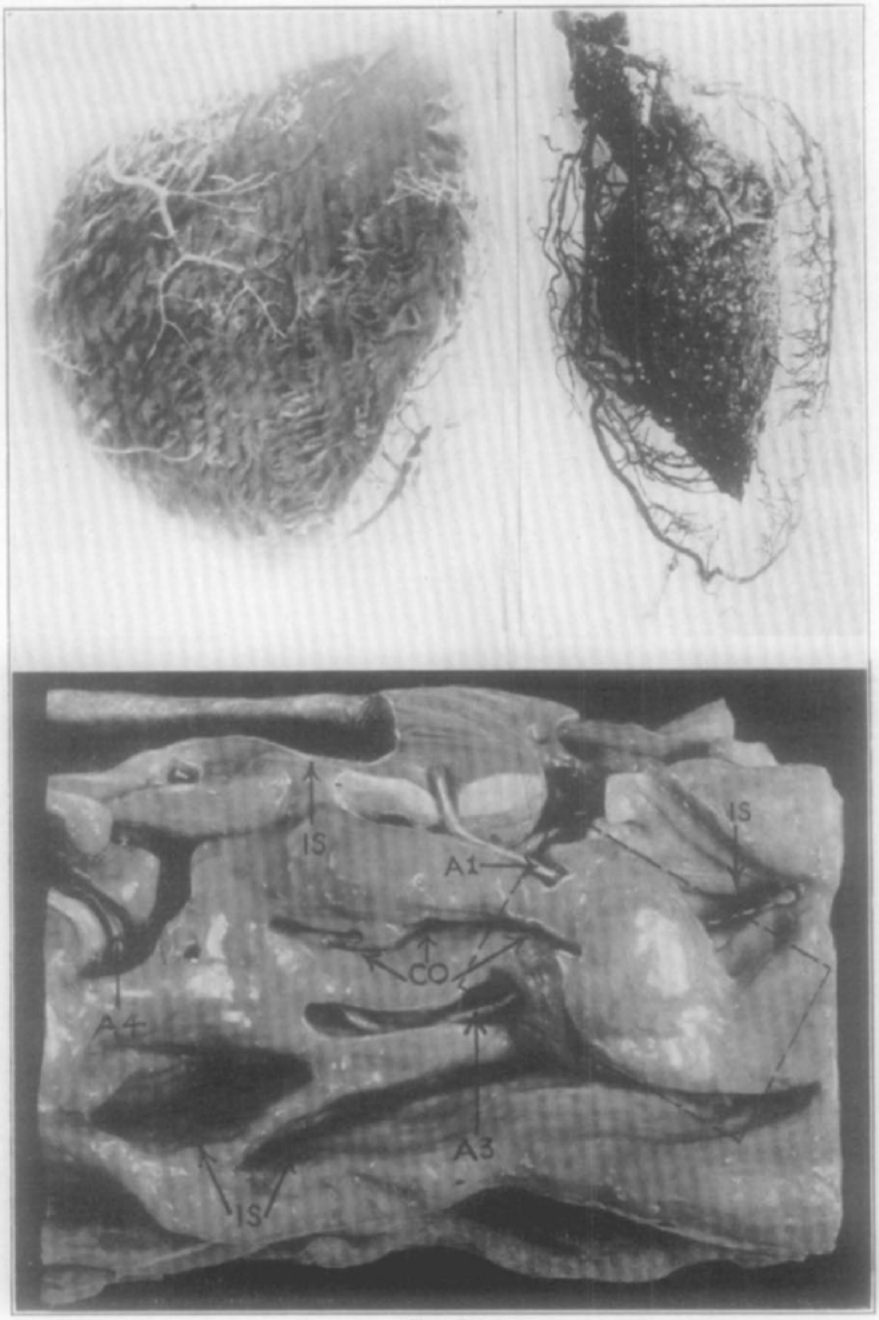


Fig. 3.

Plate II.

Fig. 1.—Heart 10. A photograph of the same specimen shown in the painting in Plate I, Figs. 1 and 2.

Fig. 2.—Heart 9. A celloidin cast of the right ventricular chamber and the coronary arteries. It shows firm fusion of the tips of some of the arteries with the cast of the chamber.

(See opposite page for explanation of Fig. 3.)

coronary artery from the base to the apex, and several openings were usually found near the apex of the ventricles. In one specimen in which both ventricles were injected, communicating vessels passed from the left anterior coronary artery to both chambers. From six to eight vessels were found with great regularity in the upper two-thirds of the anterolateral surface of the right ventricle. They have not been demonstrated with as great a frequency on the posterior surface of the heart, but in one well-injected specimen there were six entering the right ventricle posteriorly. Many of these vessels terminated not only on the surface of the columnae carneae, as represented by the depressions in Plate I, Figs. 1 and 2, and in Plate II, Fig. 1, but in the intercolumnar spaces as well.

In order to determine the relation of the arterial terminations to those of the veins a heart was prepared in the following manner:

PROTOCOL

Heart 11. Male. Aged thirty-four years. Died of pneumonia. The heart was placed in the ice box for forty-eight hours and then washed and massaged in the usual manner. A cannula was tied into each coronary artery; one in the pulmonary artery and one in the coronary sinus. The tricuspid valve was closed and made water-tight with an Ochsner clamp. A red celloidin mass was injected into the right ventricle at a pressure of 160 mm. Hg and suction was applied to the cannulas in the coronary arteries and in the coronary sinus. After fifteen minutes the suction apparatus was removed in such a manner as to prevent the entrance of air, and the blue mass was injected into the arteries and into the coronary sinus. Some of the dye escaped from the veins into the atria. Pressure was released at the end of fifteen minutes and the heart was plunged into ice water. The specimen was macerated and mounted by the technic described in the preceding protocol.

The specimen consisted of a solid red celloidin cast of the chamber of the right ventricle. Study of this specimen showed not only the red tips to the arteries as previously described, but in addition, several small red tips to the veins which were firmly fused with the blue mass in the veins, thus providing direct evidence of venous communication with the ventricular chambers. This finding of venous connections with the ventricles was in agreement with former work[6] in which serial sections demonstrated direct communication between thebesian vessels and the coronary veins.

The evidence for the existence of communicating vessels between the coronary arteries and the heart chambers in fifteen consecutive hearts

Plate II (Continued). Fig. 3.—Heart 23 Kb. A photograph of a wax-plate reconstruction of a section of the left ventricular wall near the apex, in which is shown the common opening (CO) through which several "arterio-luminal" vessels and "myocardial sinusoids" open into the lumen of the ventricle. Several intertrabecular spaces (IS) are shown. The wax has been dissected away to show the arteries (A_1, A_3, and A_4) which communicate with the cavity through the common opening. The dotted rectangle includes the tissue from which serial sections shown in Plate III, Fig. 2, and Plates IV, V, and VI were cut. Structures shown in this figure and in Plates III, IV, V, and VI are from the same heart and are designated identically for comparison. This photograph has been reduced to approximately three-fifths of the size of the original model which was constructed on a magnification of 20 times the original block of tissue.

seemed conclusive. Vieussens[1] called these vessels "vaisseaux charnus" or fleshy vessels. His reason for so designating them, however, was based upon a misconception. He believed that the myocardium was made up of vessels springing from the coronary arteries "as hairs from a wig" and he made no distinction between the vessels and the muscle fibers. For the sake of clarity and to avoid confusion, this group of vessels will henceforth be referred to in this paper as "arterio-luminal" vessels. This term is suggested inasmuch as they run from the coronary arteries into the lumen of the heart. For those connecting vessels between the coronary arteries and the atria the term "arterio-atrial" vessels might be used, and similarly for those between the coronary arteries and the ventricular chambers the term "arterio-ventricular" vessels might be employed.

Inasmuch as the structure of such vessels had never been described, the next step in the study was obvious. Moreover, it was felt that, if one of these vessels could be identified in the intact heart wall, a block of tissue containing it could be removed, sectioned serially and reconstructed. Such a study, if carried out successfully, should answer the possible criticisms that the connections shown in the celloidin casts were due to artifacts. In addition, it should establish beyond question the arterial origin of the communications and reveal the histological structure of the vessels themselves.

In approaching this task, several difficulties were encountered which necessitated a change in the injection mass employed. It was a simple matter to identify the celloidin plugs protruding from the openings of the "arterio-ventricular" communicating vessels, but, in the course of fixation, dehydration, mounting, and staining, the celloidin was dissolved and the vessels could not be traced. In order to overcome this difficulty, a suspension of Berlin blue in gelatin* was selected as an injection mass. A few experiments revealed, however, that this mass penetrated the capillaries and thus made it extremely difficult to follow the "arterio-luminal" communicating vessels. A method had to be devised, therefore, which would keep the gelatin from entering the capillaries. It was necessary of course to carry out the gelatin injections at a temperature slightly above the solidifying point of gelatin. This was accomplished by immersing the heart, injection bottle, and connecting tubing in a tank of water at the proper temperature. It was found that the introduction of cold salt solution into the ventricles at the appropriate time caused a chilling of the myocardium which allowed the gelatin to flow through the arteries and arterioles but aided in the prevention of its entrance into the capillaries. Under such conditions, it

*The injection mass was made up as follows: Soak 100 grams of Silver Label gelatin (Michigan Carbon Works) for two hours in 150 c.c. of distilled water. Warm over a water-bath until the gelatin dissolves, then add 5 grams of Berlin blue suspended in 100 c.c. of distilled water and thoroughly mix. Add 1 gram of thymol and filter the mass through several thicknesses of gauze.

was possible to inject the "arterio-luminal" communicating vessels without filling the capillary bed. When the heart was opened, solid gelatin plugs were found protruding from the endocardial openings of the channels, the gelatin having solidified on contact with the chilled salt solution in the ventricular cavities.

A block of heart muscle containing the opening, from which the gelatin protruded, was excised and placed under a binocular bi-objective dissecting microscope where the opening was identified and marked with India ink. The tissue block was then fixed and imbedded in paraffin preparatory to cutting serial sections. The following protocol illustrates the method:

PROTOCOL

Heart 23 Kb. Aged thirty-five years. Death resulted from lobar pneumonia. Heart weight 440 gm. (This weight was recorded before large clots were removed from the cavities.) This experiment was started forty hours post-mortem. Blood clots were removed from the ventricles so far as was possible. The coronary sinus and posterior great veins were tied off near their entrance to the right atrium. The coronary arteries were cannulated. Rubber tubing was led into the left and right ventricles through the aorta and the pulmonary artery respectively for the purpose of introducing chilled salt solution into these cavities. The aorta and pulmonary artery were closed by filling the space around the rubber tubing with cotton. The atrio-ventricular valves were then closed by placing cotton pledgets in their openings. These steps were taken in order to seal the ventricular chambers, thereby preventing the entrance into them of gelatin from any source other than from the openings in their own walls. The coronary cannulas and the rubber tubing connecting them with the bottle of injection mass were warmed and filled with gelatin after the removal of all air. The heart and injection apparatus were then placed in a water-bath at a temperature of 25° C. preparatory to starting the injection. The temperature of the gelatin was 53° C. Physiological salt solution chilled to a temperature of −2° C. was introduced into the ventricles in sufficient quantity to fill the chambers. After a lapse of approximately one or two minutes, the injection of Berlin blue gelatin into the coronary arteries was begun. The flow soon ceased but the injection pressure of 220 mm. Hg was maintained for about five minutes.

After sufficient time had elapsed to allow for solidification of the gelatin, the heart was opened with great care in order to prevent the entrance of any gelatin from the outside. The cotton pledgets in the atrio-ventricular valves, as well as those in the aorta and pulmonary artery, were inspected and removed. No leakage of gelatin into the ventricles had occurred.

Inspection of the myocardium showed an excellent injection of the main arteries and arterioles. That part of the heart muscle which received most of its blood supply from the left coronary artery showed some capillary injection immediately beneath the pericardium where the muscle had not been chilled. There was no evidence of capillary injection in the muscle supplied mostly by the right coronary artery.

Blocks of the heart wall showing gelatin protruding from the endocardial openings were removed from the following positions for study:

1. Right ventricle: Septal wall, halfway between pulmonary valve and apex.

2. Left ventricle: At the apex from a pocket formed by the junction of two trabeculae. (It is from this tissue that the reconstructions shown in Plate II, Fig. 3, and Plate III, Fig. 1 were made.)

The tissue removed from the left ventricle in the experiment just described was fixed, imbedded in paraffin, cut into serial sections eight micra thick, mounted and stained with van Gieson's stain. Careful study of these sections revealed convincing evidence of direct communications between the coronary arteries and the chambers of the ventricles.

Inasmuch as these communicating channels frequently ran through many of the histological sections, it was obviously impossible to publish photographs of all the sections. All sections were cut eight micra thick and for this reason sharp photomicrographs could not be obtained in each instance. It was felt, therefore, that the vessels should be reconstructed, inasmuch as such a procedure would submit our microscopic observations to a further test of accuracy. In carrying out the reconstruction, it was our good fortune to have the constant guidance and criticism of Dr. Bradley M. Patten of the Department of Histology in the School of Medicine of Western Reserve University. Dr. Patten's wide experience in this field made his advice and help invaluable.

A reconstruction in wax (Plate II, Fig. 3) was made of the block of myocardium according to the Born wax-plate reconstruction method in order to show the gross relationship of the arteries to the common opening* in the endocardium and to determine whether the common opening, into which the communicating vessels opened, connected with other intertrabecular spaces. This model was constructed on a scale twenty times the size of the original block of tissue. Its purpose was to show only gross relationships which might serve as guides in the construction of a second model of greater magnification. The wax has been dissected away in order to bring the arteries into view. Reference to the figure (Plate II, Fig. 3) will show three arteries lying in close proximity to the common opening from which the gelatin plug protruded before its removal in the process of imbedding. Branches from all three of the arteries shown opened into the common opening, and the common opening itself was found to communicate with three other intertrabecular spaces shown in the model. The lettering of the structures in the figure (Plate II, Fig. 3) is identical with that used for all other plates of the same heart (H 23 Kb) and is so arranged that the same structure is designated by the same letter in Plates III, IV, V, and VI. For instance, A_1 represents the same artery in each of these plates.

*The term "common opening" is used in the sense that Vieussens used it, namely, to indicate an inpocketing of the endocardium into which several vessels opened.

Plate III. Fig. 1.—Heart 23 Kb. Photograph of a wax-plate reconstruction of the "arteriosinusoidal" vessels and the "myocardial sinusoids" in the block of tissue shown within the broken lines in Plate II, Fig. 3. The structures shown in the model represent reconstructions of the lumina of the vessels. For instance, A_1 is an artery, AS_1 is a branch of the artery which empties directly into the common opening (CO) which is an inpocketing of the endocardium of the left ventricle. A_1 and CO, for instance, are shown in Plate II, Fig. 3, and are labeled identically. This wax-plate reconstruction was made on a scale of 200 times the original. This photograph is approximately one-fifth of the actual size of the model.

Fig. 2.—Heart 23 Kb. A photograph of one of the serial sections from which the model in Fig. I of this plate was reconstructed. The various structures in this figure are designated identically with those in Fig. 1. END = endocardium. MS = "myocardial sinusoid." CO = common opening. AS = "arterio-sinusoidal" vessel. A = artery. OMS = opening of a "myocardial sinusoid" into the lumen of the ventricle. Numbers appearing after letters enable one to identify the same structures in Plate II, Fig. 3, and Plates III, IV, V, and VI. (×41)

Fig. 1.

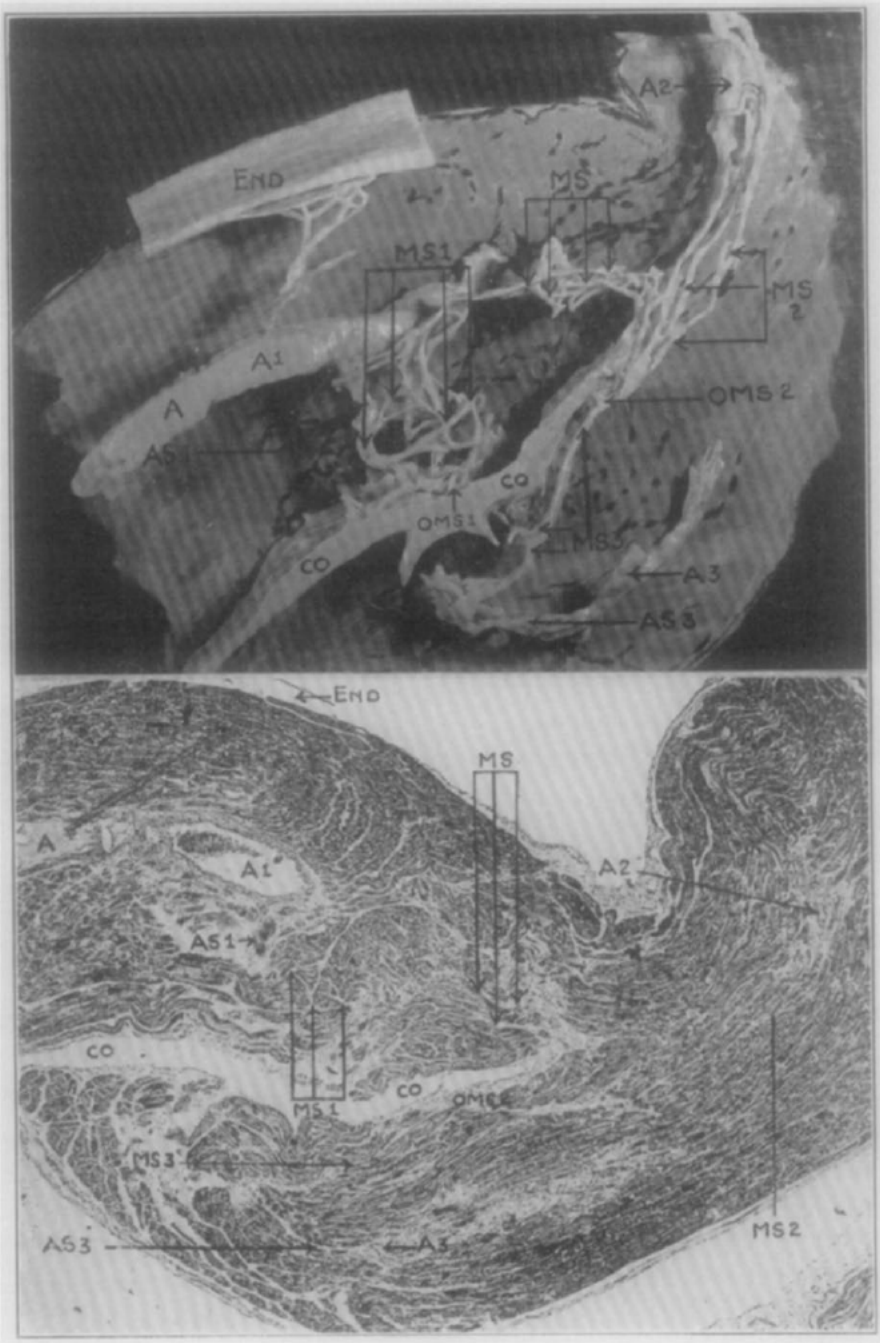


Fig. 2.

Plate III.

(See opposite page for explanation of Figs. 1 and 2.)

In one particular part of the block of myocardium which was used in making the serial sections, a number of arterial branches were found which communicated with the lumen of the ventricle via the common opening. This area is indicated by the dotted rectangle in Plate II, Fig. 3. It was found that several communicating vessels lay within this small space; consequently, it was selected for a second reconstruction. In carrying out this reconstruction, the lumina of the blood vessels and of the intertrabecular space were reconstructed so that in the final state the model represented a cast of the cavities of the blood vessels and their openings into the lumen of the ventricle through a common opening. The drawings used in the reconstruction of this model were made from projections of the individual sections at a magnification of 200 diameters. Plate III, Fig. 2 is a low power photomicrograph of one of the sections used in the reconstruction of the model shown in Plate III, Fig. 1.

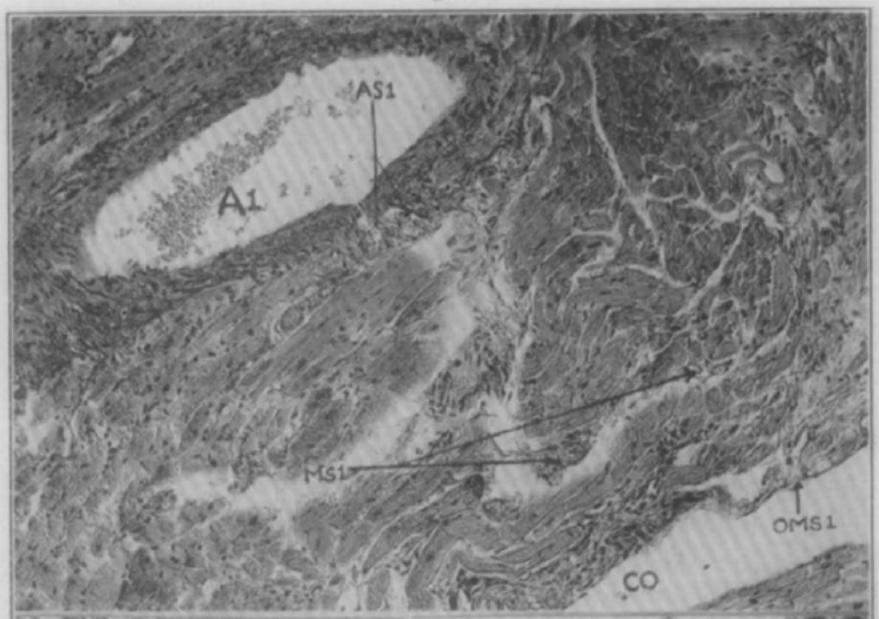
Completion of the model of the vessels furnished confirmation for our microscopic observations. Moreover, the careful study of the sections which was necessary for accurate reconstruction brought to light two distinct groups of vessels which serve as communicating channels between the arteries and the heart chambers. A vessel of the first group is usually a small branch of a coronary artery which gradually loses its arterial character through changes in its wall due to the loss of the media, thinning of the intima, and a gradual disappearance of the adventitia. Such a vessel breaks up into channels whose lumina are very irregular. The walls of these channels are very thin and are made up of endothelium only or of endothelium reinforced by a minimal amount of subendothelial connective tissue. The diameters of these channels may vary from 50 to 250 micra. The characteristics of these vessels are identical with those of sinusoids as described by Minot[15] in 1900. Their walls lie in close contact with the heart muscle, running between bundles of muscle fibers and at times between the fibers themselves. Hereafter, in this paper these channels will be referred to as "myocardial sinusoids." The arterial branches which supply the sinusoids will be referred to as the "arterio-sinusoidal" vessels. Such a name indicates the origin and the distribution of the vessels. An "arterio-sinusoidal" vessel is shown in Plate V, Figs. 1, 2, 3, and 4. A "myocardial sinusoid" is also shown in Plate V, Figs. 4 and 5. The fact that the wall of the "arterio-sinusoidal" vessel is arterial in character at the beginning and gradually changes to that of a simple endothelial tube of irregular lumen and diameter makes it inadvisable to call it either artery or vein. It can be differentiated with ease from

Plate IV. Fig. 1.—Heart 23 Kb, section 139. The branching of the artery (A_1) is shown. The branch (AS_1) divides into "myocardial sinusoids" (MS_1) which empty into the common opening (CO) at (OMS_1). See Fig. 2. (×361)

Fig. 2.—Heart 23 Kb, section 142. Shows the opening of the "myocardial sinusoid" (MS_1) into the common opening (CO). (×340)

Fig. 3.—Heart 23 Kb, section 137. Shows a section of the "arterio-sinusoidal" vessel (AS_1). The wall of the artery (A_1) from which it arises is shown.

Fig. 1.



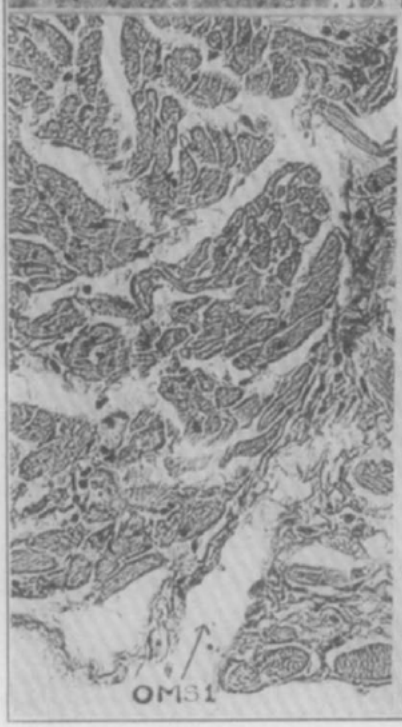


Fig. 2.

Fig. 3.

Plate IV.

(See opposite page for explanation of Figs. 1, 2, and 3.)

a capillary by its much greater diameter and by its irregular lumen. The "arterio-sinusoidal" vessels may lose their arterial character immediately after branching from the artery (Plate V, Figs. 3 and 4). At times these vessels may give off a branch which divides into capillaries.

The "myocardial sinusoids" run a meandering course, anastomose very freely with one another and not infrequently with capillaries, and open into the lumen of the ventricle either directly or through a common opening. The model (Plate III, Fig. 1) illustrates the origin of the "arterio-sinusoidal" vessels. It also shows the anastomosing "myocardial sinusoids" and their openings into the common opening which is a simple inpocketing of the endocardium (Plate III, Fig. 1). In some instances, the "myocardial sinusoids" may run a direct course from the "arterio-sinusoidal" vessel to the lumen of the ventricle. (See Plate III, Fig. 1, A2 and MS2, and Plate VI, Figs. 1 and 2.)

When closed and empty, the "myocardial sinusoids" resemble closely and are easily mistaken for strands of fibrous tissue. One frequently finds a "myocardial sinusoid" cut longitudinally with small pockets of erythrocytes here and there to identify it as a vessel. Between the pockets of erythrocytes the walls may lie against one another and resemble somewhat similar structures in the bone marrow.

The similarity of the walls of the "myocardial sinusoids" to those of capillaries and the somewhat similar distribution between, and in close contact with, muscle bundles and muscle fibers would indicate that the "myocardial sinusoids" play a definite rôle in supplying parts of the heart with blood.

The second type of communicating vessels observed ran much more directly from the coronary arteries to the lumen of the ventricle. These vessels were similar to and undoubtedly identical with the "arterio-luminal" vessels described earlier in this paper and reproduced in Plate I, Figs. 1 and 2, and Plate II, Figs. 1 and 2. The finding and identifying of the "arterio-luminal" vessels by histological methods and the tracing of their course from the artery to the heart lumen by reconstruction are confirmatory of the experiments in which celloidin injections were employed and which were described earlier in this report. Two photomicrographs of "arterio-luminal" vessels are shown in Plate

Plate V. Fig. 1.—Heart 23 Kb. section 183. Shows the branching of an "arterio-sinusoidal" vessel (AS_3) from an artery (A_3). (×410)

Fig. 2.—Heart 23 Kb, section 180. Shows the artery (A_3) and the "arterio-sinusoidal" vessel (AS_3). Note the thickness of the wall. (×372)

Fig. 3.—Heart 23 Kb, section 152. Shows the artery (A_3) at the extreme right edge of the figure and the "arterio-sinusoidal" vessel which has divided (AS_3) at the left. (×270)

Fig. 4.—Heart 23 Kb, section 137. Shows the "arterio-sinusoidal" vessel (AS_3) with the wall much thinner. At the right of the section a "myocardial sinusoid" (MS_3) is shown. (×410)

Fig. 5.—Heart 23 Kb, section 133. Shows the "myocardial sinusoid" (MS_3) into which the "arterio-sinusoidal" vessel (AS_3) opened. (×410)

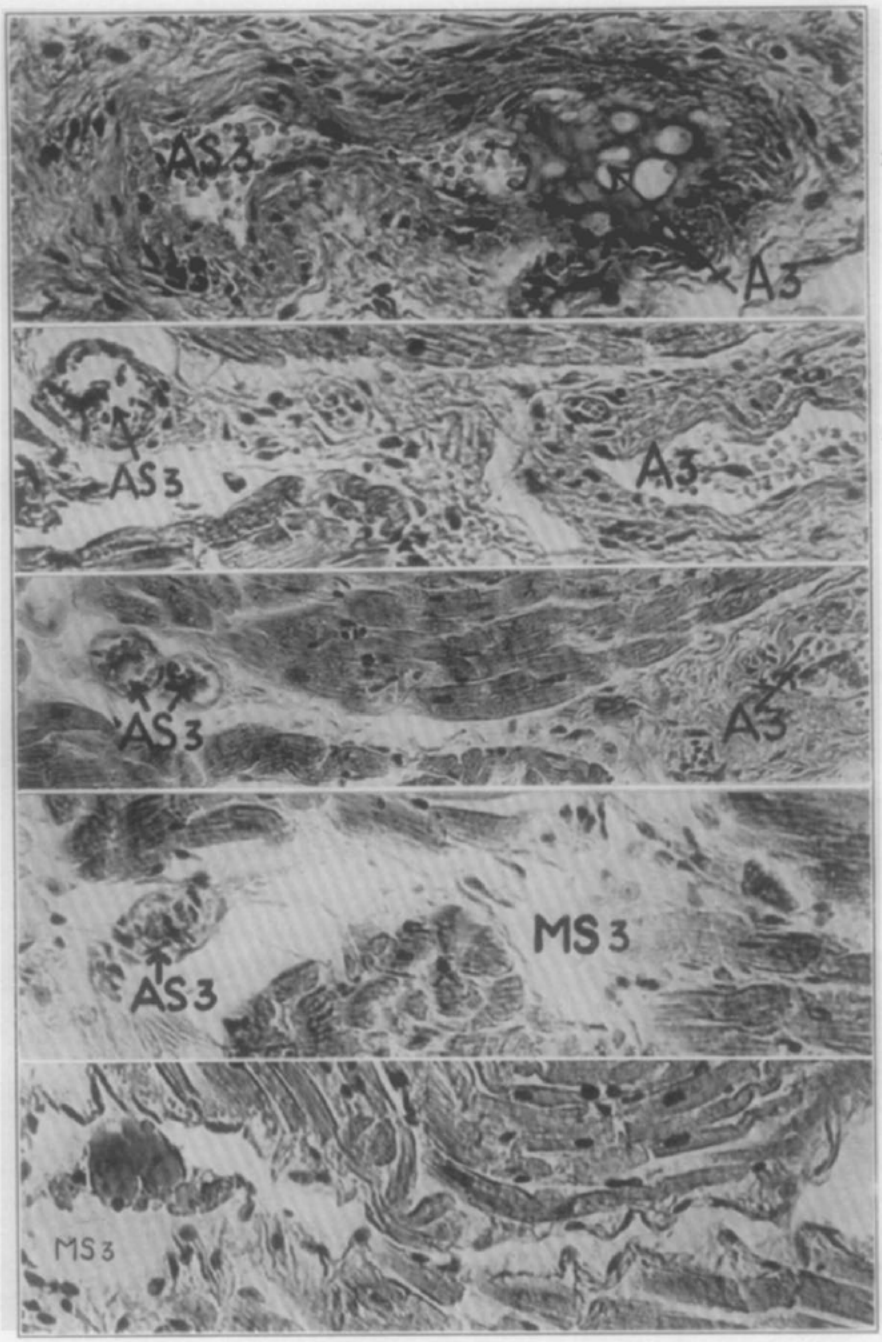


Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Plate V.

Several successive stages of an "arterio-sinusoidal" vessel (AS_3) are shown. The change in the wall from the point at which it leaves the artery (A_3) to the loss of its wall upon becoming a "myocardial sinusoid" is illustrated.

(See opposite page for explanation of Figs. 1-5.)

VII, Figs. 2 and 3, and Plate VIII, Figs. 1, 2, 3, and 4. These vessels usually run a direct, short course from the artery to the heart cavity. Even in their relatively short course, however, some of these vessels give off branches which break up into capillaries.

The walls of these vessels are thicker than those of the "arterio-sinusoidal" vessels and they retain their thickness almost to the point of entrance of the vessels into the heart cavity. At times, however, they lose their thick walls immediately after branching from the parent artery. The actual thickness of the walls or the actual diameter of the lumina is difficult to measure, since none of these vessels was found with its lumen completely filled or distended. Several diameters of vessels in the collapsed state were measured and ranged from 0.04 mm. to 0.2 mm. The diameters of the "arterio-luminal" vessels as estimated from the celloidin cast (Plate II, Fig. 1) ranged from 0.2 mm. to 1.0 mm.

The "arterio-luminal" branches of the coronary arteries were found to communicate with the atria and ventricles. They appeared to be more numerous in the ventricles than in the atria.

In view of the fact that the large number of "arterio-sinusoidal" branches and "arterio-luminal" vessels just described were observed in one small block of myocardium, a search was started for similar vessels in other hearts which had been injected by various methods.[6] The search was soon rewarded, for in the first two hearts studied "arterio-sinusoidal" vessels were found and the "myocardial sinusoids" were easily recognized. In the third and fourth hearts studied, several "arterio-luminal" vessels were discovered (Plate VII, Figs. 2 and 3, and Plate VIII, Figs. 1, 2, 3, and 4). In five consecutive human hearts studied, therefore, "arterio-luminal" or "arterio-sinusoidal" vessels were found. Inasmuch as the "myocardial sinusoids" open into the lumen of the ventricle, it follows that in tissue from five consecutive hearts from which serial sections had been prepared direct vascular communications between the coronary arteries and the heart chambers were observed.

Recent contributions have shown that the coronary circulation is not limited to the common order of vessels—artery, capillary, and vein—but is safeguarded by other channels which offer collateral routes for the blood stream. The thebesian veins, for instance, have been shown[5, 6, 10, 16] to communicate with the cardiac veins, with the capillaries, and with

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Plate VI. Fig. 1.—Heart 23 Kb, section 139. Shows artery (A_2) from which an "arterio-sinusoidal" vessel (AS_2) has just branched. The "arterio-sinusoidal" vessel (AS_2) opens into the "myocardial sinusoid" (MS_2). (×360)

Fig. 2.—Heart 23 Kb, section 139. Shows the same "myocardial sinusoid" (MS_2) as shown in Fig. 1 as it approaches the ventricular cavity via the common opening (CO). The actual opening, while not shown in this section, is in the region labeled OMS_2. At the top of the figure can be seen a closed "myocardial sinusoid" (CMS). Note the fact that MS_2 is also closed in places. (×360)

Fig. 1.

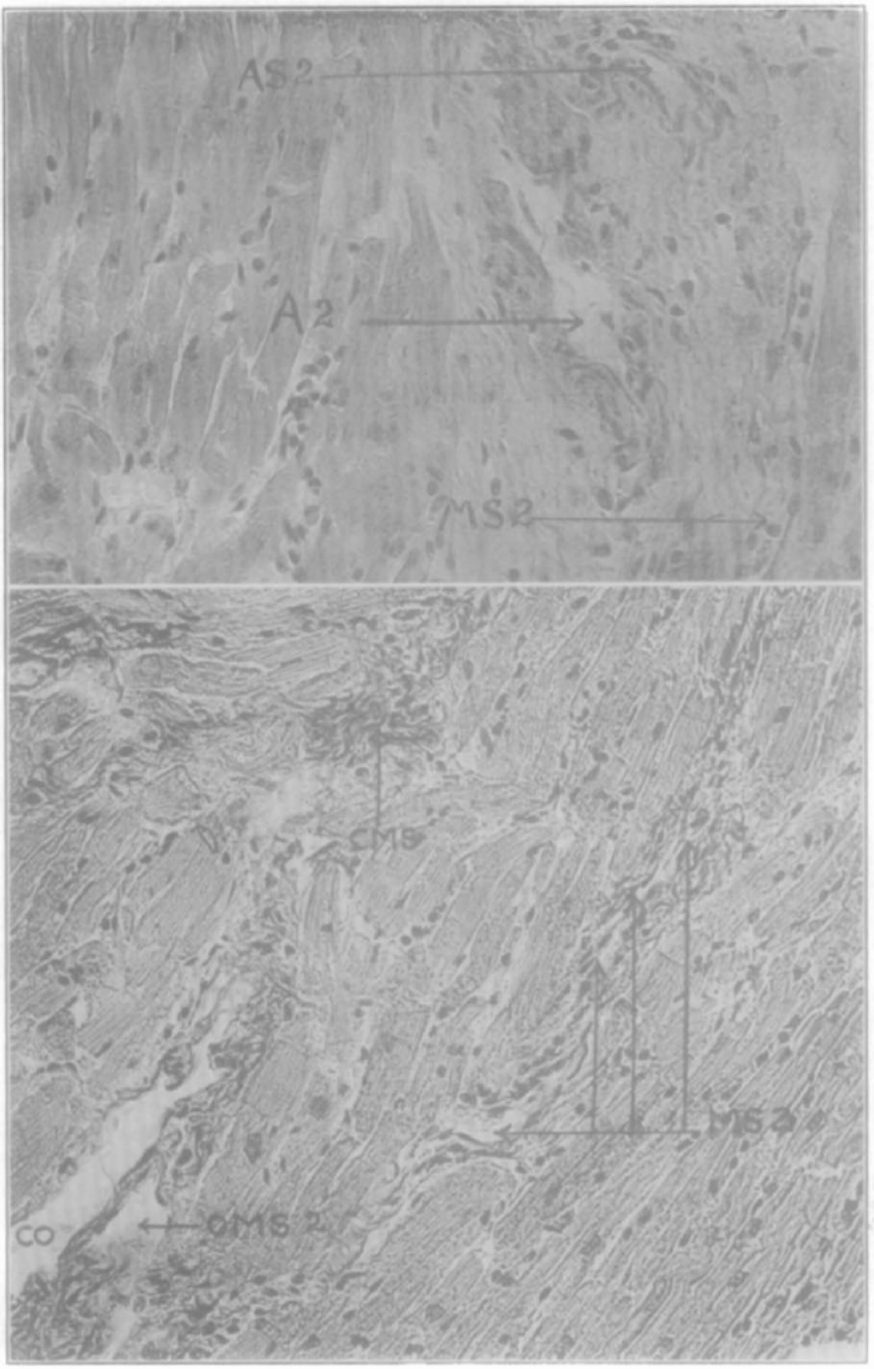


Fig. 2.

Plate VI.

(See opposite page for explanation.)

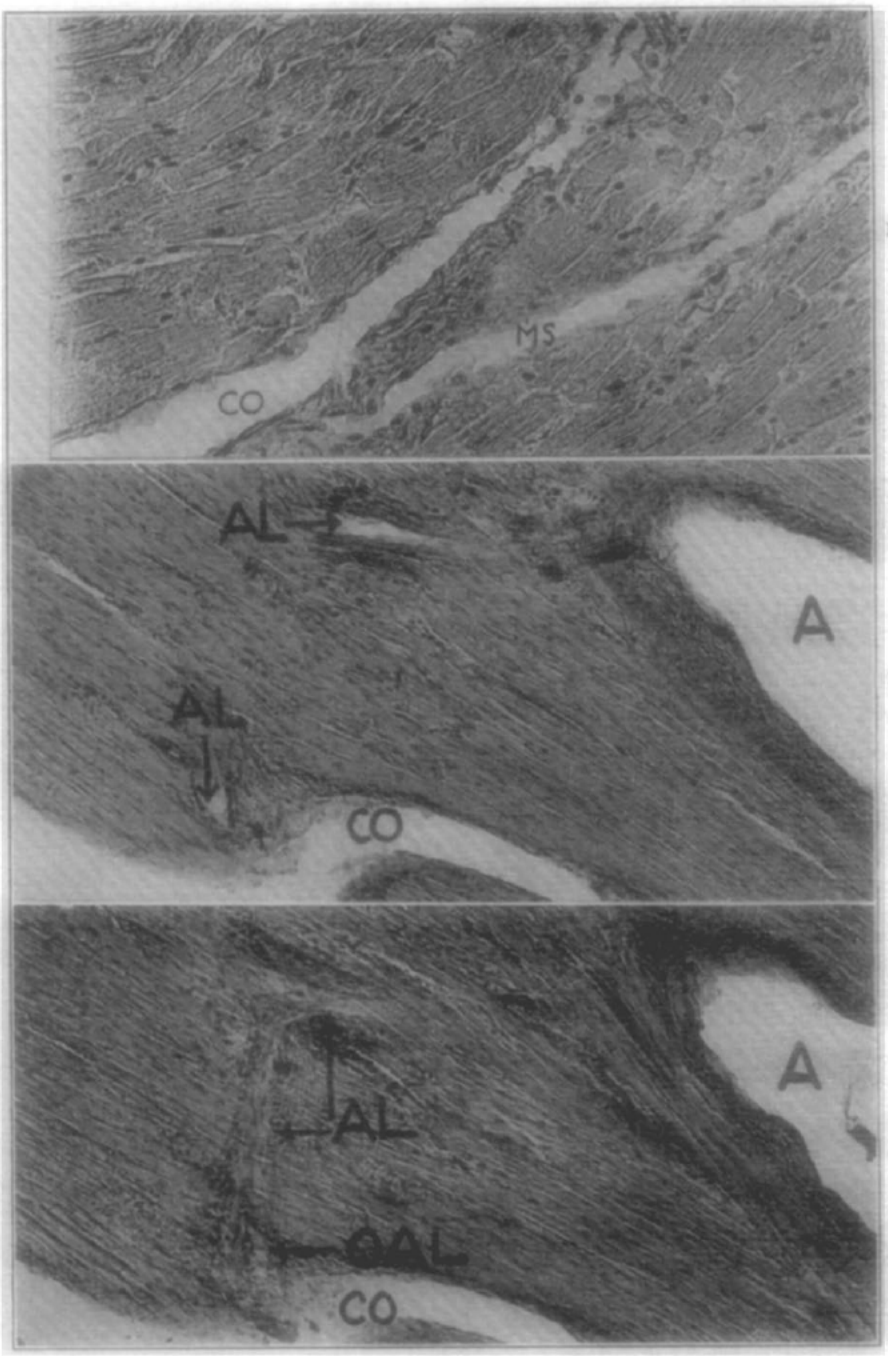


Fig. 1.

Fig. 2.

Fig. 3.

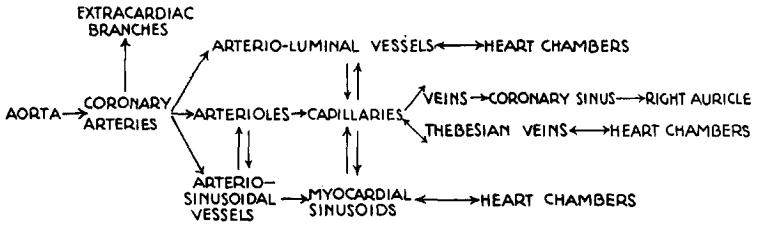
Plate VII.

Fig. 1.—Heart 23 Kb, section 142. Shows a "myocardial sinusoid" (MS) approaching the common opening (CO). (×340)

Fig. 2.—Heart 52. Shows an "arterio-luminal" vessel (AL) branching from the artery (A) and approaching the common opening (CO). Note the wall of the "arterio-luminal" vessel. (×158)

(See opposite page for further explanation of Plate VII.)

one another. More recently Hudson, Moritz, and Wearn[17] have demonstrated extensive extracardiac anastomoses of the coronary arteries which extend into the mediastinum, lungs, parietal pericardium, both surfaces of the diaphragm, and through the vasa vasorum to the abdominal aorta. The evidence presented in this paper gives additional support to the claim made by Wearn[6] in 1928; namely, that direct vascular communications other than capillaries exist between the coronary arteries and the chambers of the heart. If these various vascular channels are introduced into the scheme of the coronary circulation, it can be set up in a diagrammatic fashion as follows:



Blood entering the coronary arteries, therefore, has a possible exit through any one or all of four routes:

1. By extracardiac anastomoses;
2. By way of the capillaries and veins
   (a) into the coronary sinus or great cardiac veins and thence into the right atrium, or
   (b) through the thebesian veins into the heart chambers;
3. By way of the "arterio-luminal" vessels directly into the heart chambers; and
4. By way of the "arterio-sinusoidal" vessels through the "myocardial sinusoids" into the chambers.

The last three systems also anastomose with one another. This structural diagram is proposed in order to indicate the arrangement of the vessels and not to show the direction of the flow within them.

A somewhat similar diagram of the circulation was published by Wearn[6] in 1928, in which evidence of vascular connections between the coronary arteries and the heart chambers was presented. Inasmuch as the findings were based upon anatomical studies, no definite conclusions were drawn as to the function of the "arterio-luminal" vessels. No

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Plate VII (Continued). Fig. 3.—Heart 52. Shows the artery (A) and the "arterio-luminal" vessel (AL) which empties into the common opening (CO) at (OAL). (×158)

Figs. 2 and 3 are photographs of consecutive serial sections. A = artery. AL = "arterio-luminal" vessel. OAL = opening of "arterio-luminal" vessel into the lumen of the ventricle.

claim was made that the coronary capillaries were perfused during systole as quoted by Stella,[11] but certain speculations were indulged in which have led him to enter the field and search for the "arterio-luminal" vessels by means of a physiological method. Employing the denervated heart-lung preparation, he attempted to establish back-flow from the ventricles into the coronary arteries. Under the conditions of his experiment, the heart would beat for "one or two minutes" before beginning to fail. During these two-minute periods he was unable to establish back-flow from the chambers to the arteries and, therefore, concluded that channels between them did not exist. His results are not surprising, for it was pointed out in 1928[6] and emphasized by Leary and Wearn[18] in 1930 that, if sufficient time were allowed, the thebesian—and this should now include the "arterio-sinusoidal" vessels and the "myocardial sinusoids"—could substitute for the coronary arteries in supplying blood to the myocardium. Stella's failure to reverse the normal direction of flow and establish a back-flow in a failing heart within two minutes seems to us to have little, if any, bearing upon the existence of vascular channels between the coronary arteries and the heart chambers. Such negative evidence certainly cannot be admitted as proof that such channels do not exist.

The failure of Grant and Viko[10] to find communicating vessels between the arteries and the heart chambers led them to deny their existence. The methods employed by these workers were probably to blame for their failure. The common openings, into which the "arterio-luminal" vessels and "myocardial sinusoids" open, frequently communicate freely with the intertrabecular spaces, and this fact alone would render injection from the endocardial end almost impossible. Moreover, the openings of the "arterio-luminal" vessels usually lie deeply concealed in the intertrabecular spaces, and it is possible that Grant and Viko injected only the thebesian veins and did not find any of the "arterio-luminal" openings.

In our hands, many failures were experienced at first in attempting to inject the communicating vessels with celloidin and gelatin. The celloidin frequently solidified upon coming into contact with fluid in the arteries. The failure with the gelatin injections could be traced usually to improper temperature regulation. Careful control of these factors led to the successful injections.

Plate VIII. Fig. 1.—Heart 9B. Injection mass India ink. At the right of the figure is an artery (A) of which the "arterio-luminal" vessel (AL) is a branch. At the extreme upper left of the figure may be seen the common opening (CO). (×64)

Fig. 2.—Heart 9B. Shows a higher magnification of the "arterio-luminal" vessel (AL) and the common opening (CO) shown at the left of Fig. 1.

Figs. 2, 3, and 4 are consecutive serial sections, showing the "arterio-luminal" vessel (AL) as it approaches the common opening (CO) and then enters it at (OAL) in Fig. 4. Each figure (×247).

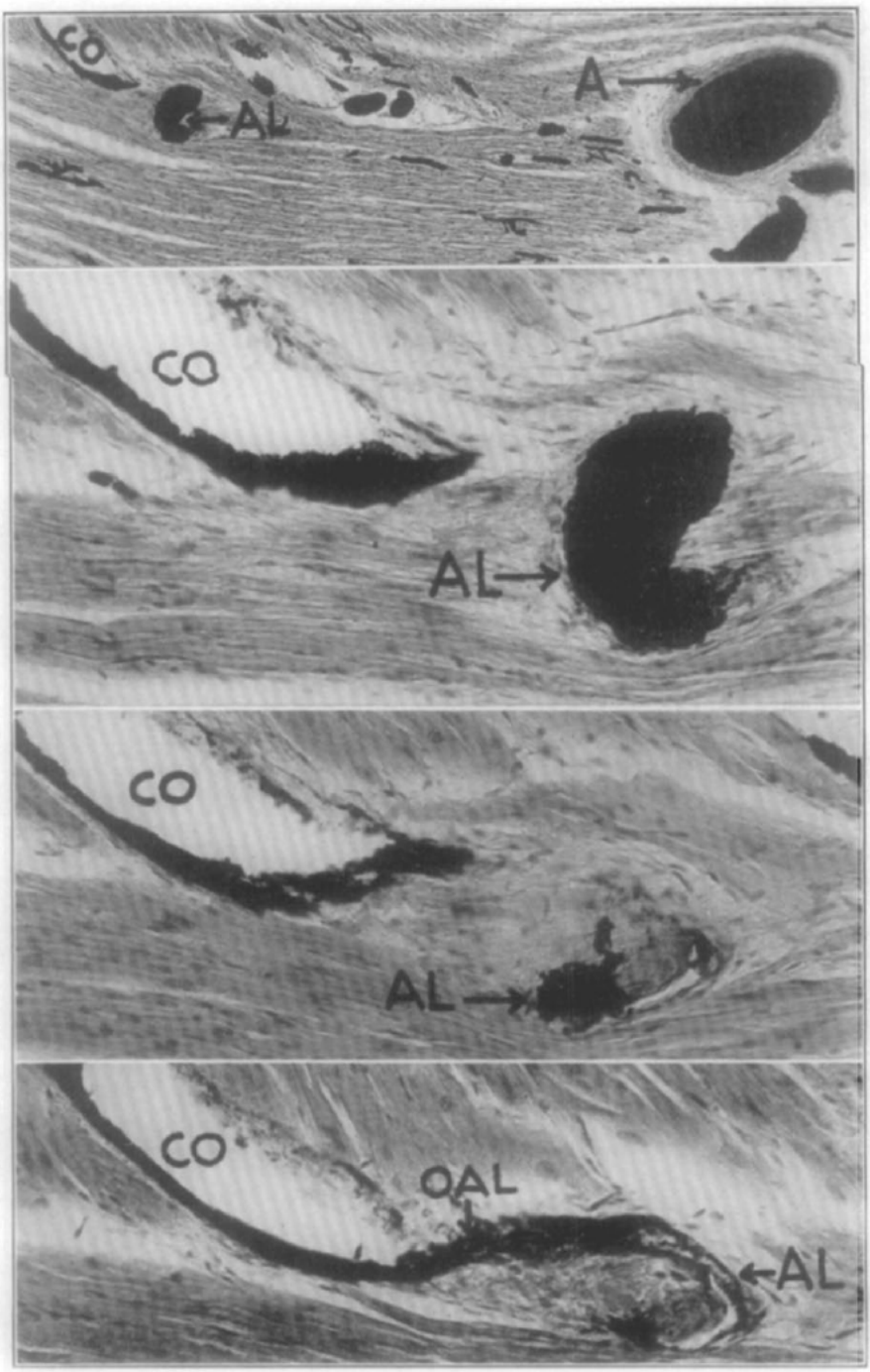

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4

Plate VIII.

(See opposite page for explanation of Plate VIII.)

SUMMARY

By the employment of injection methods, it has been possible to demonstrate vascular communications between the coronary arteries and the chambers of the heart. Serial sections and wax-plate reconstructions of these communicating vessels revealed two types which have not been described previously. The first of these communicating vessels are small branches of arteries or arterioles lying near the endocardium. They run a short course and empty directly into the lumen of the heart and, for this reason, they have been referred to as "arterio-luminal" vessels. The second type of vessel arises as a branch of an artery or arteriole and soon breaks up into sinusoids which lie between the muscle bundles and at times between the individual muscle fibers. These vessels have been referred to as "arterio-sinusoidal" vessels, and the sinusoids have been designated as "myocardial sinusoids."

The histological structure of the "myocardial sinusoids" would indicate that they play a rôle in the nourishment of the heart muscle.

It is a pleasure to express our thanks to Dr. Alan R. Moritz for his most valuable assistance in taking the photomicrographs and also for his helpful criticisms.

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